



ORIGINAL RESEARCH PAPER

Emergency Medicine

AN INTRESTING CASE OF RHABDOMYOLYSIS SECONDARY TO CEREBRAL SYPHILITIC GUMMA IN YOUNG ADULT

KEY WORDS:

Rhabdomyolysis, syphilitic gumma, VDRL, Treponema antibody

Dr. Bhargava Sai Srinivas*	Junior Resident, Department Of Emergency Medicine *Corresponding Author
Dr. Ambreesha. M	Head Of Department, Department Of Emergency Medicine
Dr. Shabbir shekhal	Associate Professor, Department Of Emergency Medicine
Dr. Souyma Subhra Datta	Assistant Professor, Department Of Emergency Medicine

ABSTRACT

Rhabdomyolysis, a potentially life-threatening condition characterized by the breakdown of skeletal muscle tissue and release of intracellular contents, commonly occurs in response to trauma, prolonged immobilization, and certain medications(1). However, its occurrence secondary to neurological disorders, such as cerebral syphilitic gumma, is exceedingly rare(3). Cerebral syphilitic gumma, a tertiary manifestation of syphilis, presents as granulomatous lesions in the brain, typically causing neurological symptoms. The co-occurrence of rhabdomyolysis with syphilitic gumma, though unusual, may reflect the multifactorial impact of neuromuscular compromise such as seizures, prolonged immobilization, and metabolic disturbances triggered by central nervous system pathology(8). We report a case of a 24-year-old male who presented with complaints of involuntary movements of bilateral upper and lower limbs, headache, and difficulty in walking. Laboratory findings revealed elevated creatinine kinase levels (1239 mcg/L), myoglobinuria (>3000 ng/mL) and creatinine 4.2mg/dl leading to initial diagnosis of rhabdomyolysis. MRI of the brain showed a well-defined, ring-enhancing lesion in the right frontal lobe. VDRL serology was reactive and treponema antibody positive. Diagnosis was revised to rhabdomyolysis with acute kidney injury secondary to cerebral syphilitic gumma. Patient was stabilized in ED and treated with benzathine penicillin. Patients with tertiary syphilis poses risk for neurological complications like, focal neurological deficits and convulsions rhabdomyolysis. The incidence of tertiary syphilis has declined in developed countries, but untreated cases still pose a risk for such rare manifestations. Early recognition and appropriate management in ED are crucial to prevent severe systemic complications in these patients.

INTRODUCTION

Rhabdomyolysis is a potentially life-threatening condition characterized by the breakdown of skeletal muscle tissue, leading to the release of intracellular contents, including myoglobin, into the bloodstream(1). Rhabdomyolysis is commonly associated with trauma, prolonged immobilization, certain medications or toxins and secondary to neurological disorders causing convulsions(2).

Cerebral syphilitic gumma, a manifestation of tertiary syphilis, presents as localized granulomatous lesions in the brain, causing a spectrum of neurological symptoms such as headache, altered mental status, focal neuro deficits and convulsions(7). The incidence of cerebral syphilitic gumma has declined in developed countries due to widespread antibiotic use, but it remains a potential diagnosis in untreated or inadequately treated cases of syphilis(8). On the other hand, the incidence of rhabdomyolysis is more frequent, particularly in association with traumatic or metabolic triggers.

However, the involvement of muscle breakdown, as seen in rhabdomyolysis, is highly uncommon in the context of tertiary syphilis. Although both conditions are individually rare, their co-occurrence may reflect the multifactorial impact of neuromuscular compromise, prolonged immobilization, and metabolic disturbances triggered by the underlying central nervous system pathology(8). Documented cases of rhabdomyolysis secondary to cerebral syphilitic gumma are extremely scarce in medical literature. This case report highlights the need for thorough clinical assessment and diagnostic consideration in patients presenting with tertiary syphilis.

Case Study

A 24 years old male presented to emergency department with complaints of involuntary movements of bilateral upper and lower limbs 2 episodes since one day associated with frothing from the mouth, and complaints of headache since one day. On arrival he was conscious, oriented to time, place and person with Pulse rate 100bpm, Blood pressure of 124/88 mmHg, Saturation 97% at room air, Respiratory rate of 17per minute, body temperature 98.5°F and GRBS-113mg/dl.

On secondary survey CNS examination revealed tandem gait. Examination of other systems was unremarkable. Indurated ulcer was noticed over shaft of penis during foleys catheterization with a low volume cola coloured urine output. Patient was admitted to emergency ward and standard treatment was started. Urine myoglobin showed greater than 3000ng/ml , creatinine 4.2mg/dl and CPK 1239mcg/lit. Rhabdomyolysis was suspected. Intravenous crystalloid increased to 300ml/hr. ABG was normal. Ultrasound abdomen finding was increased renal cortical echogenicity and 2D ECHO was normal. Initial NCC'T of brain revealed edema over right frontal region.

Patient underwent MRI BRAIN with contrast on day2 of admission suggestive of well defined round mildly lobulated ring enhancing lesion appearing T1 hypointense with hyperintense rim, T2 hypointense rim in the right frontal lobe with adjacent perilesional edema suggestive of space occupying ring enhancing lesion likely tuberculoma or syphilitic gumma or fungal abscess (fig 1,2)., Serology showed VDRL-reactive. treponema antibody showed increased titres to 24(normal less than 0.1)

At this stage diagnosis was revised as rhabdomyolysis secondary to convulsions caused by cerebral syphilitic gumma. Patient was continued intravenous crystalloids

300ml/hr with antiepileptics, and antiedema measures. Antibiotics changed from ceftriaxone to benzathine penicillin 2.4 million units IM. On day4 serum creatinine was 1.4mg/dl. ABG was normal, Urine output was normal. Patient was transferred to general medicine for further follow up.

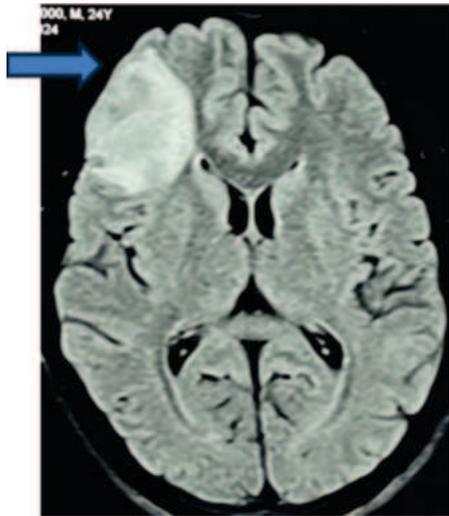


Fig-1-T2 hyperintense with hypointense rim in the right frontal lobe with adjacent perilesional edema.

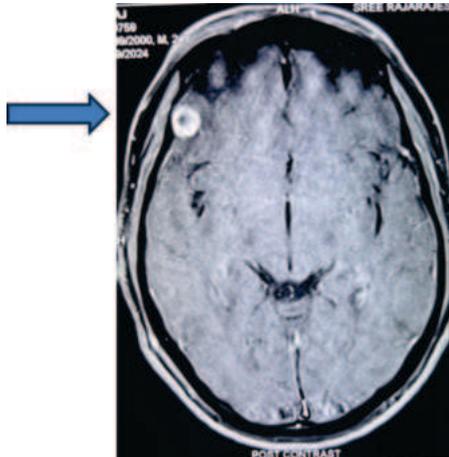


Fig-2- Well defined round mildly lobulated enhancing lesion appearing T1 hypointense with hyperintense rim.

DISCUSSION:

Rhabdomyolysis is a clinical syndrome characterized by the breakdown of damaged skeletal muscle tissue, leading to the release of intracellular components like creatine kinase, myoglobin, and electrolytes into the bloodstream(1). This process can result in systemic complications such as acute kidney injury (AKI) and electrolyte imbalances(1).

The condition may be caused by a variety of factors, including traumatic injuries, intense physical exertion, prolonged immobilization, infections, and certain medications(2). It is also associated with metabolic myopathies, extreme temperatures, and substance abuse (e.g., alcohol or cocaine). Non-traumatic causes include statin use, severe infections, and autoimmune diseases like polymyositis(2).

Clinically, rhabdomyolysis is often identified by a classic triad of symptoms: muscle pain, weakness, and dark urine due to myoglobinuria(3). However, these symptoms are present in only about 10% of cases, making a high index of suspicion crucial for diagnosis. Laboratory evaluation showing elevated serum creatine kinase levels (typically >5 times the normal upper limit) is key to diagnosis.

Treatment primarily focuses on preventing kidney damage

through aggressive hydration, electrolyte management, and addressing underlying causes. Early detection and intervention are critical to improving outcomes(3).

In India, syphilis remains a significant public health issue, particularly among underserved populations(5). Although the overall incidence of tertiary syphilis has decreased, cases of syphilitic gummas still surface, particularly in individuals with poor access to healthcare. Rhabdomyolysis secondary to syphilitic gumma is extremely rare(6).

One hypothesis is that chronic granulomatous inflammation in the muscles may lead to local ischemia, necrosis, and eventual muscle breakdown(7) Tertiary syphilis often presents with neurological symptoms, the association with muscle damage such as rhabdomyolysis remains poorly understood (8).

Early recognition of this unusual presentation is crucial for avoiding severe complications such as acute renal failure. The diagnosis is confirmed through serological testing, imaging, and histopathological examination of the lesions (9). Delayed treatment or misdiagnosis is common in resource-limited settings, contributing to more severe presentations.

Treatment with appropriate antibiotics, such as penicillin 2.4 million units is essential for resolving the underlying infection and preventing further muscle damage(11).

CONCLUSIONS

This case highlights the importance of maintaining clinical suspicion for early recognition of tertiary syphilis in young patients presenting with neurological symptoms. Comprehensive care can mitigate the risk of severe outcomes such as acute kidney injury and permanent neurological deficits.

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